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A REVIEW ON POLYCYSTIC OVARY SYNDROME (PCOS)

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ABSTRACT

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age and is associated with reproductive, metabolic, and psychological complications. Its diverse presentation makes diagnosis and management challenging, highlighting the importance of a comprehensive understanding of its mechanisms and outcomes. This review begins with an overview of the symptoms and normal menstrual cycle and contrasts it with the hormonal disturbances that characterize PCOS, including anovulation and altered ovarian morphology. Clinical features such as irregular cycles, hirsutism, infertility, and the presence of polycystic ovaries are discussed, along with metabolic abnormalities, particularly insulin resistance, which is recognized as a central factor linking reproductive and metabolic dysfunction. Management strategies are explored across lifestyle, nutritional, and pharmacological domains. Dietary modification, supplements such as inositol and vitamin D, and herbal agents like cinnamon are reviewed for their potential benefits, while conventional therapies including oral contraceptives and metformin remain widely used. Overall, PCOS requires individualized, multidisciplinary care that integrates lifestyle, medical, and supportive approaches.

KEYWORDS: *Polycystic Ovary Syndrome (PCOS), Menstrual cycle, Hyperandrogenism, Hirsutism, Infertility, Management, Combined Oral Contraceptives (COC).*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrinopathy affecting women of reproductive age. Common features include menstrual irregularities, hyperandrogenism and polycystic ovarian morphology although the presentation can be heterogeneous [1]. Polycystic ovary syndrome (PCOS) typically manifests with a combination of menstrual dysfunction and hyperandrogenism in the adolescent population. It is associated with derangements in insulin secretion and action, androgen synthesis and action, relative gonadotropin ratios, ovulatory function, and balance of pro- and antioxidant systems [2]. Polycystic ovary syndrome (PCOS) is characterized by hyperandrogenism and chronic anovulation, often leading to infertility in affected women. Globally, approximately 68% of women with PCOS present with elevated androgen levels, menstrual irregularities, and multiple small ovarian cysts. In 2003, an international consensus group identified the clinical manifestations of hyperandrogenism to include acne, hirsutism, dyslipidemia, insulin resistance, type 2 diabetes mellitus, obesity, reproductive dysfunction, and an increased risk of cardiovascular disease [3].

Insulin resistance is a central feature of PCOS, occurring even in lean women, and may differ mechanistically from obesity-related insulin resistance. Women with PCOS are more likely to experience overweight, obesity, and greater long-term weight gain compared to those without PCOS. Weight gain and obesity worsen insulin resistance, amplifying PCOS symptoms. Weight management—through prevention of weight gain, modest weight loss, or maintenance—is considered a first-line treatment in international guidelines. Effective strategies require multidisciplinary lifestyle interventions, including dietary changes, physical activity, and behavioral therapies [4].

Most symptoms of PCOS arise early during puberty. Since PCOS involves a combination of signs and symptoms, thus it is considered as a heterogeneous disorderliness. The most accepted diagnostic criteria are Rotterdam criteria which involves two of the latter three features: (a) hyperandrogenism, (b) oligo- or an-ovulation, and (c) polycystic ovaries. The persistent hormonal imbalance leads to multiple small antral follicles formation and irregular menstrual cycle, ultimately causing infertility among females [5]. Unhealthy lifestyle, diet or any infectious mediators increase the risk of PCOS. Due to insulin resistance and its elevated level, the ovaries' function disturbs the androgen level which leads to anovulation (11). Apart from the environmental factors, there are genetic factors that are responsible for the etiology of PCOS [6].

Polycystic Ovary Syndrome [PCOS]

Polycystic ovarian syndrome (PCOS) is a common endocrine disorder affecting 8–13% of women of reproductive age. It is characterized by anovulation and dysfunction of the hypothalamic–pituitary–ovarian axis, distinguishing it from other ovulation failures.

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Women with PCOS often face risks such as endometrial hyperplasia, insulin resistance, metabolic syndrome, and chronic low-grade inflammation.

Recent advances have improved understanding of its pathophysiology, diagnosis, and treatment. Current approaches emphasize:

- Lifestyle modifications (diet, exercise, weight management)
- Type 2 diabetes medications to address insulin resistance
- Bariatric surgery for severe metabolic complications

The broader page context highlights PCOS not only as a reproductive disorder but also as a metabolic condition, with prevention and management strategies targeting long-term health risks [7].

Multifactorial Nature of PCOS: PCOS is not caused by one factor alone; instead, it results from interactions between genes, gene–gene relationships, and gene–environment influences. This complexity makes it difficult to pinpoint a single cause or marker.

Genetic Predisposition: Research highlights the importance of hereditary factors in PCOS development. However, no consensus exists on a definitive genetic marker that can reliably predict or diagnose PCOS. Scientists are working to identify causal variants— specific changes in genes that may alter protein function and contribute to the disorder [2]. Polycystic ovary syndrome (PCOS) is consistently characterized by elevated testosterone or androstenedione levels, and recent studies highlight its complex biological underpinnings. Research shows that insulin resistance and hyperandrogenism interact to drive PCOS development, while androgen excess can damage mitochondrial structure in granulosa cells, impairing ovarian function. Genetic and molecular findings include ANGPTL4 overexpression linked to metabolic disorders, altered calcium entry contributing to hormonal imbalance, and identification of DLGAP5 as a candidate gene. Neurological factors also play a role, with epilepsy and antiseizure medications affecting PCOS through the hypothalamic–pituitary–ovarian axis. Immune and inflammatory mechanisms further complicate the condition: IL-15 influences granulosa cell survival and steroid production, and adipose-derived extracellular vesicles carrying miR-26b promote cell apoptosis. Altogether, these studies emphasize that PCOS is a multifaceted disorder involving metabolic, genetic, immune, and neurological pathways that converge to disrupt ovarian function and fertility [8].

Symptoms of PCOS

Menstrual Irregularities

- Infrequent, irregular, or absent menstrual cycles (oligomenorrhea or amenorrhea).

Infertility

- Difficulty conceiving due to anovulation or hormonal imbalance.

Weight-Related Issues

- Overweight or obesity, often linked to insulin resistance.

Hormonal Disturbances Associated

Reproductive Hormones

- Elevated luteinizing hormone (LH).
- Altered follicle-stimulating hormone (FSH).
- Increased LH/FSH ratio.
- Disrupted gonadotropin-releasing hormone (GnRH) pulsatility

Sex Steroids

- Hyperandrogenism (elevated androgens like testosterone).
- Estrogen and progesterone imbalance.

Metabolic Hormones

- Insulin resistance and hyperinsulinemia.
- Altered levels of ghrelin, LEAP2.

Growth Hormone Axis

- Possible dysregulation of growth hormone (GH) [9].

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Menstrual irregularities manifest as infrequent or heavy periods, amenorrhea, and dysfunctional uterine bleeding, while infertility and subfertility are commonly observed. Additional symptoms may include acne, excessive hair growth, and hair loss. Light brown, velvety hair on the neck, armpits, and groin, as well as the presence of a "string of pearls" small cysts in the ovary, may also be observed [10].
Normal Menstrual Cycle:

Reproductive Lifespan

- Women typically have ~36 years of reproductive activity, from menarche (8.5–13 years) to menopause (~51 years).
- Puberty lasts ~2.3 years, ending with menarche.

Cycle Length and Phases

- The "textbook" cycle is 28 days, but actual cycles vary widely (25–34 days).
- Follicular phase averages ~14.6 days; luteal phase ~13.6 days in women aged 19–42.
- Variability is greatest in the follicular phase (10–23 days), which shortens with age.
- Only ~10% of women with a 28-day cycle have perfectly balanced 14-day follicular and luteal phases.

Age-Related Changes

- Cycle length shortens after age 35, with irregularities near menarche and menopause.
- Anovulation (absence of ovulation) occurs in ~7% of women aged 25–39 with normal cycles, but is much higher in adolescents (60% of girls aged 10–14) and older women (34% over age 50).

Menstrual Bleeding

- Bleeding marks the transition from luteal to follicular phase.
- Typically lasts 3–6 days (range 2–12), with heaviest flow on Day 2.
- Average blood loss is ~33 ml (range 10–84 ml).
- With age, bleeding duration shortens slightly, but blood loss increases (older women lose ~6 ml more than younger women).

Influences and variability

Regional, ethnic, and socioeconomic factors may affect cycle length, phase duration, and bleeding patterns [11].

Etiopathogenesis

Polycystic Ovary Syndrome (PCOS) is a complex, multifactorial disorder with a significant genetic component, although its precise etiology remains unclear. To elucidate the underlying pathophysiology, various animal models have been employed, each with inherent limitations. Rodents are the most extensively utilized, followed by mice, rhesus monkeys, ewes, and more recently, sheep. These models have contributed valuable insights, particularly highlighting the role of intrauterine exposure to excess androgens in predisposing individuals to PCOS later in life.

The genetic basis of PCOS has been further substantiated through familial studies. Legro et al. investigated the siblings of 80 women diagnosed with PCOS and found that among 115 sisters, 46% exhibited hyperandrogenemia, with nearly half meeting the diagnostic criteria for PCOS. Additionally, male siblings demonstrated elevated levels of dehydroepiandrosterone sulfate (DHEAS) compared to age-matched controls, suggesting a broader familial endocrine dysregulation. Parental phenotypes also reflect this predisposition: fathers of adolescent girls with PCOS show a markedly high prevalence of obesity (94%) and metabolic syndrome (79%), while mothers tend to be either obese (54.4%) or overweight (11.4%), with 34% exhibiting metabolic syndrome. These findings collectively underscore the heritable and systemic nature of PCOS, reinforcing the importance of genetic and intrauterine factors in its pathogenesis [12].

Mechanism/ Pathophysiology:

The pathophysiology and intrinsic mechanisms under lying PCOS are complex because etiologies vary and the different features are considerably intertwined(fig.2). The interplay between these mechanisms results in and perpetuates the clinical features of PCOS, including hyperandrogenism, PCOM and ovulatory dysfunction, in addition to the associated mood disturbances, psychosexual dysfunction and long-term morbidities. In addition, the development of PCOS has a strong genetic component [13].

PCOS diagnosis lacks a single definitive test and relies on clinical evaluation and exclusion of other endocrine disorders such as thyroid disease, hyperprolactinemia, Cushing's syndrome, and adrenal hyperplasia. Recommended assessments include pelvic examination, transvaginal ultrasound, hormone profiling, and detailed medical history focusing on insulin resistance and menstrual irregularities. The

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most widely accepted diagnostic framework is the Rotterdam criteria, which require the presence of at least two of the following: hyperandrogenism, ovulatory dysfunction, or polycystic ovarian morphology [14].

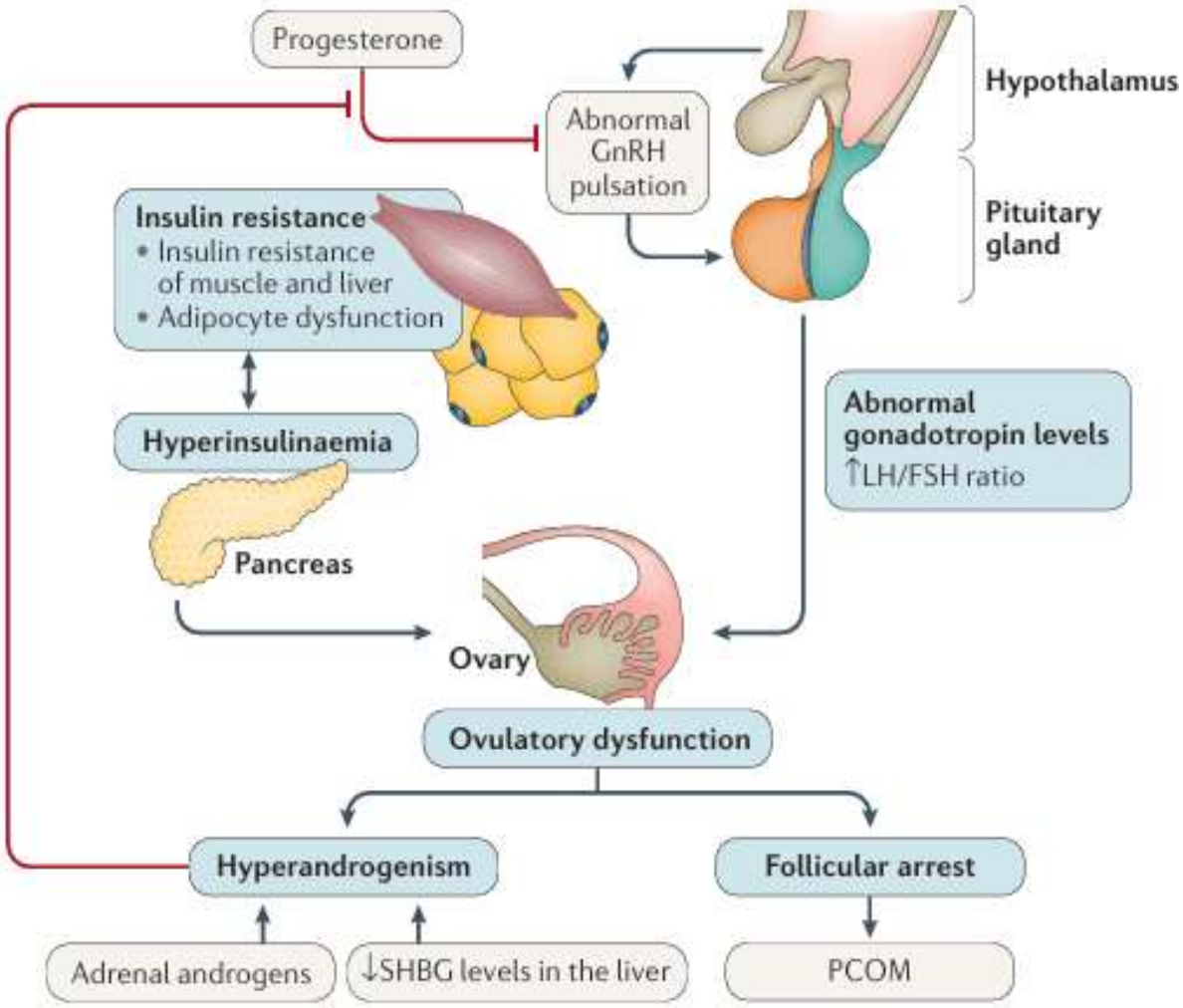


Figure 1 | *The pathophysiology of PCOS.*

The pulsatile release of gonadotropin- releasing hormone (GnRH) from the hypothalamus is often disturbed in polycystic ovary syndrome (PCOS), leading to luteinizing hormone (LH) hypersecretion by the pituitary gland, which induces ovulary dysfunction and hyperandrogenism. This perturbed secretion of LH seems to arise early in puberty and is related to disturbed inhibition of GnRH secretion by progesterone. Although serum follicle-stimulating hormone (FSH) levels are generally normal, follicles seem to be more resistant to FSH in women with PCOS than in controls. This effect might be due to increased levels of intra-ovarian anti-Müllerian hormone (AMH). Notably, genetic and epigenetic variants contribute considerably to susceptibly for most of these alterations. Environmental factors contribute somewhat less, most by exacerbating insulin resistance and dysregulated gonadotropin secretion. PCOM, polycystic ovarian morphology; SHBG, sex hormone-binding globulin [13].

Hyperandrogenism and Hirsutism:

Androgen excess is a central feature of polycystic ovary syndrome (PCOS), affecting approximately 60% to 80% of individuals with the condition. This hormonal imbalance manifests clinically as hyperandrogenism, which is the most prevalent abnormality observed in PCOS and contributes significantly to its pathogenesis. Common symptoms include hirsutism, acne, seborrhea, and androgenic alopecia, all of which stem from elevated androgen levels [15]. Hirsutism is a remarkable clinical symptom of hyperandrogenism common in PCOS which can be visually diagnosed. It is a major consequence of androgen excess. Hyperandrogenism leads to the presence of

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masculine features in the females with PCOS. The presence of excessive facial hairs on the side of the face, upper lip, chin and it is also observed in chest region in individuals with severe conditions of PCOS. Hirsutism is defined by the FG score (Ferriman - Gallwey) though it has certain limitations [16].

Insulin Resistance:

Insulin Resistance (IR) or hyperinsulinemia stimulates the theca cells of the ovary and acts synergistically with luteinizing hormone to produce excessive testosterone, which is responsible for the clinical symptoms of hyperandrogenism (acne, hirsutism, alopecia). Insulin also inhibits hepatic synthesis of sex hormone-binding globulin and thus increases the proportion of free testosterone while the total testosterone concentration is at the upper range of normal or only modestly elevated [17]. Between 50 and 70% of women with polycystic ovary syndrome (PCOS) exhibit demonstrable insulin resistance, which provokes a compensatory increase in insulin secretion by the pancreatic β-islet cells. Despite this adaptive response, many women with PCOS also experience impaired β-cell function, and the severity of this dysfunction appears to be influenced by a family history of type II diabetes. The resulting state of hyper insulinemic insulin resistance not only exacerbates metabolic disturbances but also stimulates ovarian androgen production, thereby worsening the endocrine abnormalities characteristic of PCOS. This dual impact—on both metabolic and reproductive pathways—significantly increases the likelihood of developing type II diabetes and contributes to the broader spectrum of long-term health risks associated with the syndrome [18].

It is important to note that the disturbed regulation of insulin in the central nervous system has been connected to obesity and poor ovarian follicular maturation, which points toward more links between obesity and PCOS [15].

Polycystic Ovaries on Ultrasound:

Ultrasound is the premier extensively used technique for the ultrasound examination of PCO. The sonographic criteria have been thusly balanced and, along these lines, the addition in ovarian volume (>10 cm3) and the proximity of >12 follicles with a broadness of 2 to 9 mm at smallest in one ovary.

In extension to these criteria, other restorative conditions that can cause steady an ovulation and androgen excess should be restricted [3].

Polycystic Ovaries

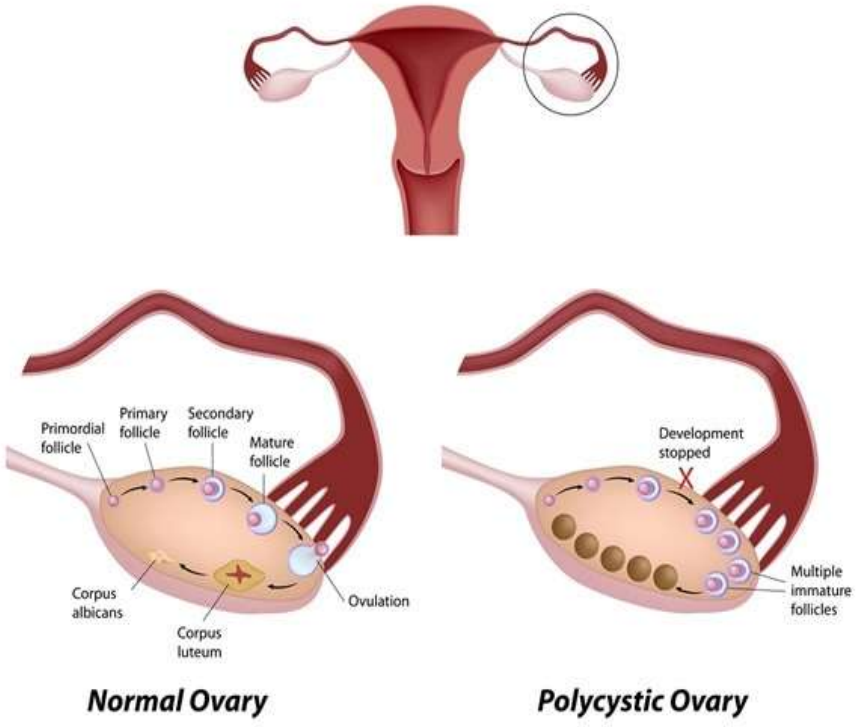


Figure 2: Normal ovarian cycle and Polycystic ovary syndrome.

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Polycystic ovary syndrome (PCOS) is distinguished by the presence of multiple small ovarian cysts that develop due to an imbalance between follicle growth and ovulation. These cysts, typically less than 10 mm in size, often appear as a "string of pearls" on ultrasound and usually resolve spontaneously within a few cycles. Unlike neoplastic ovarian cysts, they are lined by a simple layer of flat or cuboidal epithelium, reflecting their less complex morphology. Importantly, PCOS cysts are hormonally active, producing excess androgens, estrogens, and progestogens, which contribute to hallmark symptoms such as menstrual irregularities, hirsutism, and acne. Their presence is also closely linked to insulin resistance, obesity, and metabolic syndrome, underscoring the broader endocrine and metabolic implications of PCOS beyond reproductive health [10].

Oxidative stress & inflammation in PCOS:

- Women with PCOS show elevated markers of lipid peroxidation.
- Increased **C-reactive protein (CRP)** and inflammatory cytokines.
- Higher concentrations of circulating lymphocytes and monocytes.
- These findings suggest a systemic inflammatory state [19].

Infertility: Polycystic ovarian syndrome (PCOS) is recognized as the most common cause of anovulation, accounting for infertility in up to 80% of affected women. Its prevalence is estimated to range between 9–18% among reproductive-age women, though this varies depending on the diagnostic criteria applied. The Rotterdam criteria remain the most widely accepted framework, requiring the presence of at least two of the following features: clinical or biochemical evidence of hyperandrogenism, oligo- or anovulation, and polycystic ovarian morphology on ultrasound. Beyond reproductive dysfunction, PCOS is frequently associated with obesity and metabolic syndrome, reflecting underlying insulin resistance and dyslipidemia. Psychological comorbidities, including anxiety and depression, are also common, further complicating the clinical picture. Importantly, infertility in PCOS may arise not only from anovulation but also from impaired oocyte quality and reduced endometrial receptivity, underscoring the multifaceted nature of this syndrome and the need for comprehensive, individualized management strategies [20].PCOS is the first cause of anovulatory infertility nowadays and infertility is found in 70 to 80% of affected women [21].

Genetic Basis Of PCOS

Large-scale genome-wide association studies (GWAS) have only consistently identified a handful of genetic variants across populations. This indicates that while genetics play a role, they are not the sole determinant. PCOS is highly heterogeneous, meaning genetic predisposition may interact differently with environmental factors in different individuals [22].

Evidence accumulated over several decades indicates a strong familial aggregation of polycystic ovary syndrome (PCOS), hyperandrogenism, and related metabolic disturbances, supporting a genetic basis for the disorder. Although the precise inheritance model remains undefined, PCOS is most commonly regarded as a polygenic condition, with multiple susceptibility loci contributing to disease expression. In certain families, a single gene may exert a predominant influence on phenotype. Candidate genes include those involved in androgen biosynthesis, transport, regulation, and receptor activity, as well as genes associated with insulin metabolism, such as the insulin receptor and downstream signaling proteins. These findings underscore the multifactorial nature of PCOS, wherein genetic predisposition interacts with metabolic and endocrine pathways to produce heterogeneous clinical manifestations [23].

Psychological impact of pcos: Since 2014, great attention has been paid to the comorbidities shown in PCOS [16]. PCOS women show increased odds of depressive and anxiety symptoms [24].

Fatigue and sleep disturbances are among the most frequently reported depressive symptoms in women with polycystic ovary syndrome (PCOS), and their presence may serve as indicators of underlying depression. Evidence consistently demonstrates that both the prevalence and severity of depressive symptoms are elevated in women with PCOS compared to controls. A recent meta-analysis encompassing 57 studies and 172,040 participants found that women with PCOS had 2.79 times greater odds of receiving a clinical diagnosis of depression relative to women without PCOS. Similarly, another meta-analysis estimated the prevalence of depression in this population to be approximately 36%. Although higher body mass index (BMI) has been implicated as a potential risk factor, its association with depression in PCOS appears relatively weak. Instead, the pathophysiology of depression in PCOS may be more strongly linked to endocrine and metabolic disturbances, including hyperandrogenism, hyperinsulinemia, and elevated inflammatory markers, all of which may contribute to the development and exacerbation of depressive symptoms [4].

Screening for Psychological Wellbeing: Women diagnosed with polycystic ovary syndrome (PCOS) should undergo systematic screening for psychological disturbances at the time of diagnosis. Current clinical guidelines emphasize the importance of evaluating not only depression and anxiety, but also negative body image, disordered eating, and psychosexual dysfunction. Positive screening results warrant further clinical assessment and referral to appropriate specialists. Emerging evidence also highlights an elevated risk of depression among adolescents with PCOS, underscoring the need for targeted psychological screening in this population [17].

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Alteration of Hormones in PCOS:

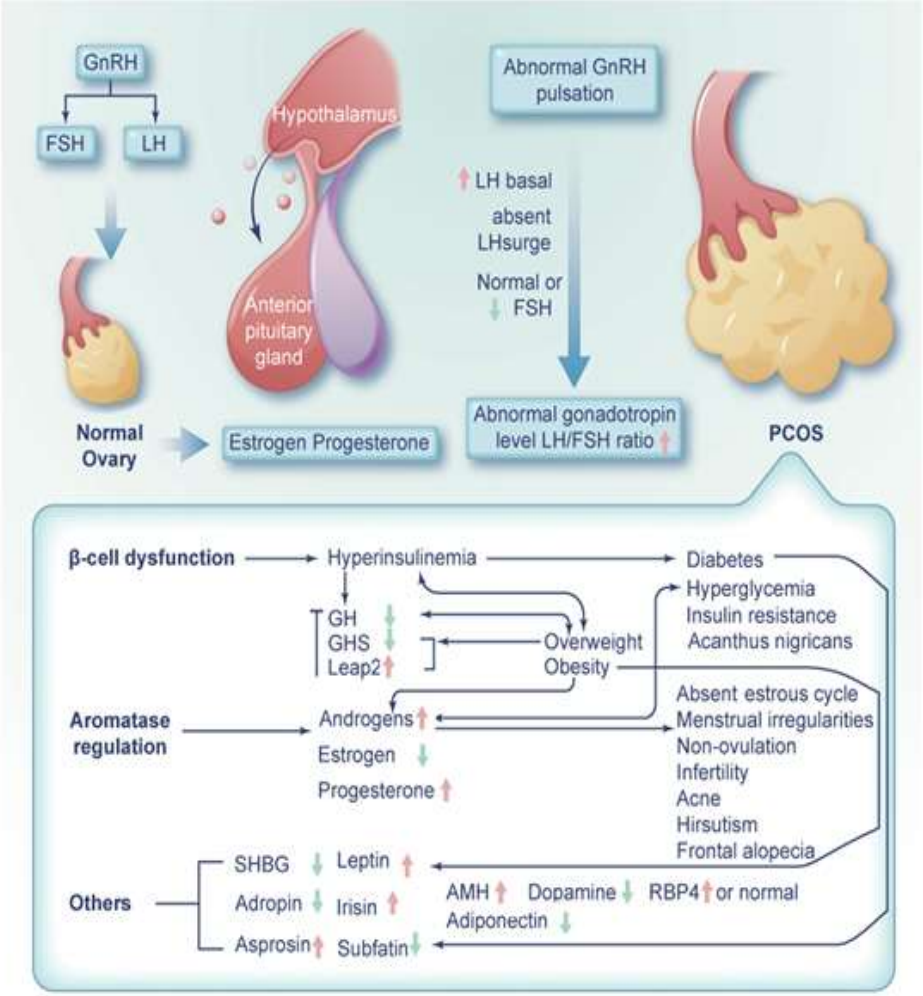


Fig 3: Systematic representation of various hormonal alterations in PCOS. Reprinted from [9].

Diagnosis:

Multiple clinical conditions can mimic lean PCOS clinically and need exclusion by appropriate laboratory work up before planning management. A diagnostic algorithm for work up of suspected cases of PCOS is shown in Fig 4 [1].

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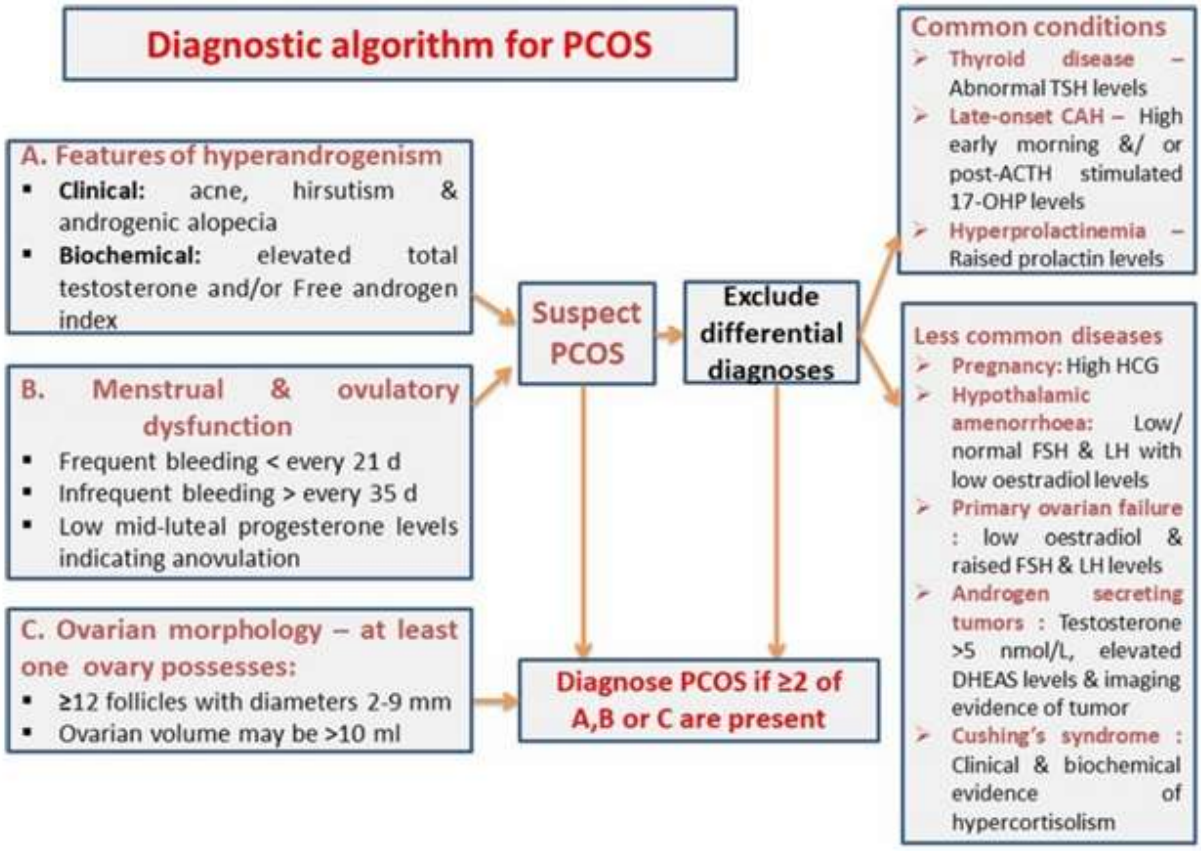


Fig. 4: A diagnostic algorithm for polycystic ovary syndrome (PCOS). TSH– thyrotrophin, ACTH–adrenocorticotrophin, 17-OHP– 17 hydroxyprogesterone, HCG human chorionic gonadotrophin, FSH– follicle stimulating hormone, LH– luteinizing hormone, DHEAS dehydroepiandrosterone sulphate [1].

Management of PCOS

Polycystic ovary syndrome (PCOS) involves numerous systemic and metabolic factors beyond the ovaries, yet the core disturbance lies in ovarian dysfunction. Ongoing debates persist around both its diagnostic criteria and the most effective approaches to treatment [25]. The choice of management strategy and therapy in PCOS must be tailored to the individual patient and her specific priorities. Clinical concerns may range from achieving fertility and regulating menstrual cycles to reducing weight or alleviating hyperandrogenic manifestations such as acne, hirsutism, and androgenic alopecia [26].

PCOS Management: Beyond Medication

Diet and Lifestyle modification

PCOS patients are primarily recommended to reduce weight since a good, balanced diet combined with regular exercise can raise their metabolism, improve insulin sensitivity, and help them lose weight safely, since over half of all PCOS sufferers are overweight or obese [27]. Lifestyle modifications by dietary interventions and regular physical activity have demonstrated improved insulin resistance and ameliorated hyperandrogenism amongst other beneficial effects on PCOS symptoms. Lean individuals with PCOS must be encouraged to consume vegetables and fruit to ensure they are having an adequate supply of various minerals, vitamins and nutrients [1].

The 2018 international guideline on PCOS highlights that current evidence does not support the superiority of any specific dietary approach for improving health outcomes. Recommendations emphasize adopting balanced dietary strategies tailored to individual lifestyle needs and preferences, consistent with general population guidance. Systematic reviews comparing dietary compositions such as low carbohydrate, low glycemic index/load, high protein, MUFA-enriched, and fat-counting diets reveal minimal differences in anthropometric outcomes, with weight loss itself being the primary determinant of improvement in PCOS features. Emerging research,

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however, suggests that certain dietary strategies may exert beneficial effects on PCOS manifestations independent of weight reduction. These findings warrant careful consideration to inform both clinical practice and consumer decision-making [28].

Impact of Omega-3 fatty acids

Omega-3 fatty acids have been identified as beneficial in the management of polycystic ovary syndrome (PCOS). Their biological properties include anti-inflammatory, antithrombotic, antiarrhythmic, and antiatherogenic effects, which contribute to improved cardiovascular and metabolic health. In women with PCOS, omega-3 intake has been associated with reductions in hirsutism, body mass index (BMI), luteinizing hormone (LH), testosterone, insulin, and sex hormone-binding globulin (SHBG) levels, thereby supporting menstrual cycle regulation and lipid profile improvement.

Primary dietary sources include fish oil, as well as plant-based options such as chia, flax, and perilla seeds. Beyond PCOS-specific outcomes, omega-3 fatty acids confer broader protective effects against cardiovascular disease, cancer, neurological, and hormonal disorders, largely attributable to their antioxidant activity. Supplementation may serve as an adjunct strategy when dietary intake is insufficient [29].

Vitamin D : In 2014, Lerchbaum and colleagues studied how vitamin D affects reproduction. They found that women with normal vitamin D levels had thicker endometrium, which made pregnancy more likely. While the exact way vitamin D helps women with PCOS is not yet understood, newer studies suggest that taking vitamin D3 can be beneficial. It may reduce the harmful effects of advanced glycation end products (AGEs), support hormone balance, and improve the growth of ovarian follicles in women with PCOS [30].

Exercise: Engaging in at least 150 minutes of moderate-intensity exercise per week**,** as recommended by the Physical Activity Guidelines for Americans, helps improve cardiometabolic health and manage PCOS symptoms. Simple activities like walking, swimming, or cycling are effective ways to meet this goal [10].

Sleep Modification: Psychological issues play a dual role as both risk factors and sustaining elements of illness in adolescents and young women with PCOS. Research shows that sleep disorders are the most common psychological comorbidity, and because they influence PCOS development, sleep management is a crucial component of lifestyle interventions for affected females [22].

Inositol: The isomers myo-inositol and D-chiro-inositol play complementary roles in the treatment of PCOS in the liver, with myo-inositol promoting glucose uptake and D-chiro-inositol increasing glycogen synthesis [31]. According to a new study, menstrual periods and ovulation can be improved. Although this recommendation cautions against using Inositol owing to the limited advantages, it also has a low risk of adverse effects and is cheap [7]. Myo-inositol, a natural insulin sensitizer, has been extensively investigated in women with polycystic ovary syndrome (PCOS), particularly in the context of assisted reproduction. In a small randomized controlled trial, Papaleo et al. demonstrated that supplementation with myo-inositol combined with 2 g per day of folic acid reduced gonadotropin requirements during ovarian stimulation and resulted in fewer immature oocytes.

Building on these findings, a meta-analysis of eight studies confirmed a consistent reduction in both gonadotropin dose and stimulation duration among PCOS patients undergoing IVF. Similarly, Zheng et al. conducted a meta-analysis of seven trials, four of which included PCOS patients, and reported that pretreatment with myo-inositol was associated with higher clinical pregnancy rates alongside lower gonadotropin use. Collectively, these data suggest that myo-inositol improves ovarian response efficiency, reduces pharmacologic burden, and enhances reproductive outcomes in PCOS patients undergoing IVF [20].

Vitamin E: Vitamin E (or tocopherol) is a fat-soluble vitamin that can be stored in the liver and released in small quantities to maintain physiological levels. Vitamin E exhibits antioxidant properties as it neutralizes free radicals and promotes cell renewal. Recent evidence has confirmed that vitamin E may improve endometrial thickness in women with idiopathic infertility, thanks to its anticoagulant and antioxidant properties [30].

Herbal Treatment:

Herbal treatment for PCOS also include use of various natural herbs also, such as *Cinnamon Cassia, Tribulus Terrestris, and* Triphala (*Emblicaofficinalis, Terminaliabeletica, Terminaliachebula)* [32].

Cinnamon Cassia: The natural herb, Dalchini, has shown a sensitizing effect in pre-clinical and clinical studies. The polyphenols present in the herb show an effect on increasing insulin-dependent glucose metabolism and, thus, the glucose transport is altered. The study suggests that women with PCOS can be treated with Dalchini as it improves irregularity in the menstrual cycle and provides effective treatment without side effects [32].

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The new therapeutic tools

Insulin sensitizers

The strong pathophysiological connection of insulin resistance with PCOS aberrations sup ports the therapeutic use of insulin sensitizers in the management of PCOS. The extensive literature has shown that reduction in insulin levels pharmacologically ameliorates the sequelae of hyperinsulinemia and hyperandrogenemia. Insulin sensitizers, mainly metformin and thiazolinediones, can effectively manage the established metabolic derangements in PCOS, but whether they can prevent them is not yet established [33].

Metformin

Metformin is a biguanide medication that has been proven to be both safe and effective. Even though it is still an authorized application, metformin has long been used to treat type 2 diabetes and is one of the most often utilized insulin sensitizers in treating PCOS. Metformin improves insulin sensitivity in peripheral tissues by lowering hepatic glucose production, boosting glucose absorption, and reducing hepatic glucose synthesis. Metformin side effects include nausea, vomiting, diarrhea, and abdominal distension. In PCOS, dyslipidemia treatment is critical. Metformin reduces dyslipidemia by directly reducing hyperinsulinemia or altering the liver's free fatty acid metabolism. Metformin is prescribed to women with PCOS at a beginning dose of 500-850 mg per day, which can be raised to 2000 mg per day if tolerated. Metformin in higher doses can help people lose weight and improve their lipid profiles, especially if they are obese and have PCOS. Metformin usage over a long period has also been associated with vitamin B12 deficiency [27].

Combined Oral Contraceptives (COC)

COCs are combinations of low-dose estrogens and progestins. Combined oral contraceptives (COCs) exert their contraceptive effect primarily through the actions of both progestin and estradiol.

The progestin component directly inhibits gonadotropin-releasing hormone (GnRH) secretion, thereby suppressing the luteinizing hormone (LH) surge that is essential for ovulation. In the absence of this LH peak, ovarian sensitivity to follicle-stimulating hormone (FSH) is reduced, leading to diminished estradiol production. Progestins also contribute to contraceptive efficacy by increasing the viscosity of cervical mucus, which impedes sperm penetration, while simultaneously decreasing tubal motility and thinning the endometrial lining to prevent implantation. The estradiol component complements these effects by suppressing the FSH surge, thereby inhibiting the selection and maturation of a dominant follicle. In addition, estradiol stabilizes the endometrium, maintaining its proliferation and improving menstrual cycle control, which reduces the incidence of breakthrough bleeding. Together, these mechanisms ensure effective prevention of ovulation, fertilization, and implantation while promoting cycle regularity [34]. While there is a big controversy regarding the effects of oral contraceptives on adiposity indices, it is well documented that all oral contraceptives, regardless of their progestin type, can improve menstrual regularity, mainly through suppress hypothalamus pituitary gonadal [35].

CONCLUSION

This review highlights the complex and multifactorial nature of polycystic ovary syndrome (PCOS), emphasizing its impact on reproductive, metabolic, and psychological health. Hormonal dysregulation disrupts the normal menstrual cycle, leading to anovulation, polycystic ovarian morphology, and clinical manifestations such as hirsutism and infertility.

Insulin resistance emerges as a central mechanism linking metabolic and reproductive dysfunction, underscoring its importance in disease progression. Management strategies span lifestyle modification, dietary regulation, nutraceuticals including inositol and vitamin D, herbal agents such as cinnamon, and conventional therapies like oral contraceptives and metformin. Collectively, these approaches demonstrate the need for individualized, multidisciplinary care. Future research should refine diagnostic criteria and evaluate long-term therapeutic outcomes to improve patient-centered management and enhance quality of life for women affected by PCOS.

REFERENCES

1. *S. Toosy, R. Sodi, and J. M. Pappachan, "Lean polycystic ovary syndrome (PCOS): an evidence-based practical approach," Journal of Diabetes & Metabolic Disorders, vol. 17, no. 2, pp. 277–285, Nov. 2018, doi: 10.1007/s40200-018-0371-5.*
2. *Muhammad Jaseem Khan, Anwar Ullah & Sulman Basit (2019) Genetic Basis of Polycystic Ovary Syndrome (PCOS): Current Perspectives, The Application of Clinical Genetics, 249-260, DOI: 10.2147/TACG.S200341.*
3. *Miss Dipamoni Morang | Pankaj Chasta | Mr. Kaushal K. Chandrul "A Review on "Polycystic Overy Syndrome (PCOS)"" Published in International Journal of Trend inScientific Research and Development (ijtsrd), ISSN: 2456 6470, Volume-3 | Issue-4, June 2019, pp.60-66, URL: https://www.ijtsrd.c om/papers/ijtsrd23 542.pdf.*
4. *C. Ee, S. Pirotta, A. Mousa, L. Moran, and S. Lim, "Providing lifestyle advice to women with PCOS: an overview of practical issues affecting success," BMC Endocrine Disorders, vol. 21, no. 234, pp. 1–12, 2021, Doi: 10.1186/s12902-021-00890-8.*
5. *S. Siddiqui, S. Mateen, R. Ahmad, and S. Moin, "A brief insight into the etiology,genetics, and immunology of polycystic ovarian syndrome (PCOS)," Journal of Assisted Reproduction and Genetics, vol. 39, pp. 2439–2473, Oct. 2022,doi: 10.1007/s10815-022-02625-7.*

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 12 | December 2025 - Peer Reviewed Journal

6. T. Piri-Gharaghie, "Polycystic ovary syndrome and genetic factors influencing its development: A review article," Personalized Medicine Journal, vol. 6, no. 23,pp. 25–29, Autumn 2021, doi: 10.22034/pmj.2021.249631.
7. S. Akre, K. Sharma, S. Chakole, and M. B. Wanjari, "Recent advances in the management of polycystic ovary syndrome: A review article," Cureus,vol. 14, no. 8, p. e27689, Aug. 2022, doi: 10.7759/cureus.27689.
8. Y. Wang, P. Leung, R. Li, Y. Wu, and H. Huang, "Editorial: Polycystic ovary syndrome(PCOS): Mechanism and management," Frontiers in Endocrinology, vol. 13,Art. no. 1030353, Oct. 2022, doi: 10.3389/fendo.2022.1030353.
9. J. Yang and C. Chen, "Hormonal changes in PCOS," Journal of Endocrinology,vol. 261, no. e230342, pp. 1–20, Feb. 2024, doi: 10.1530/JOE-23-0342.
10. H. Bai, H. Ding, and M. Wang, "Polycystic Ovary Syndrome (PCOS): Symptoms,Causes, and Treatment," Clin. Exp. Obstet. Gynecol., vol. 51, no. 5, p. 126, May 2024, doi: 10.31083/j.ceog5105126.
11. M. Mihm, S. Gangooly, and S. Muttukrishna, "The normal menstrual cycle in women,"Animal Reproduction Science, vol. 124, no. 3–4, pp. 229–236, Sep. 2010.doi: 10.1016/j.anireprosci.2010.08.030.
12. N. Madnani, K. Khan, P. Chauhan, and G. Parmar, "Polycystic ovarian syndrome,"Indian Journal of Dermatology, Venereology, and Leprology, vol. 79, no. 3,pp. 310–321, May–Jun. 2013. doi: 10.4103/0378-6323.110759.
13. R. Azziz, E. Carmina, Z. Chen, A. Dunaif, J. S. E. Laven, R. S. Legro, D. Lizneva, B. Natterson-Horowitz, H. J. Teede, and B. O. Yildiz, "Polycystic ovary syndrome," Nature Reviews Disease Primers, vol. 2, no. 16057, pp. 1–18, Aug. 2016. doi: 10.1038/nrdp.2016.57.
14. M. Di Lorenzo, N. Cacciapuoti, M. S. Lonardo, G. Nasti, C. Gautiero, A. Belfiore,B. Guida, and M. Chiurazzi, "Pathophysiology and Nutritional Approaches inPolycystic Ovary Syndrome (PCOS): A Comprehensive Review," Current Nutrition Reports, vol. 12, no. 4, pp. 527–544, May 2023. doi: 10.1007/s13668-023-00479-8.
15. S. V. Waghmare and A. Shanoo, "Polycystic Ovary Syndrome: A Literature ReviewWith a Focus on Diagnosis, Pathophysiology, and Management," Cureus, vol. 15,no. 10, p. e47408, Oct. 2023. doi: 10.7759/cureus.47408.
16. S. Abinaya, D. Siva, R. Sabitha, and S. Achiraman, "An overview of hyperandrogenismin PCOS and the prospective underlying factors," Research Journal of Life Sciences, Bioinformatics, Pharmaceutical and Chemical Sciences, vol. 5, no. 1, pp. 179–186, Jan. 2019. doi: 10.26479/2019.0501.18.
17. S. Shermin, A. Noor, and S. Jahan, "Polycystic Ovary Syndrome: A Brief Review with Recent Updates," Delta Medical College Journal, vol. 7, no. 2, pp. 84–99, Jul. 2019.doi: 10.3329/dmcj.v7i2.47344.
18. A. S. Laganà, P. Rossetti, M. Buscema, S. La Vignera, R. A. Condorelli, G. Gullo, R. Granese, and O. Triolo, "Metabolism and ovarian function in PCOS women:A therapeutic approach with inositols," International Journal of Endocrinology,vol. 2016, Article ID 6306410, pp. 1–9, 2016. doi: 10.1155/2016/6306410.
19. 19. R. Pasquali, E. Stener-Victorin, B. O. Yildiz, A. J. Duleba, K. Hoeger, H. Mason, R. Homburg, T. Hickey, S. Franks, J. S. Tapanainen, A. Balen, D. H. Abbott, E. Diamanti-Kandarakis, and R. S. Legro, "PCOS Forum: research in polycystic ovarysyndrome today and tomorrow," Clinical Endocrinology, vol. 74, no. 4, pp. 424–433,Apr. 2011, doi: 10.1111/j.1365-2265.2010.03956.x.
20. A. M. Kotlyar and D. B. Seifer, "Women with PCOS who undergo IVF: a comprehensivereview of therapeutic strategies for successful outcomes," Reproductive Biology and Endocrinology, vol. 21, no. 70, pp. 1–9, Jul. 2023, doi: 10.1186/s12958-023-01120-7.
21. J. Collée, M. Mawet, L. Tebache, M. Nisolle, and G. Brichant, "Polycystic ovarian syndrome and infertility: overview and insights of the putative treatments," Gynecological Endocrinology, vol. 37, no. 10, pp. 869–874, Oct. 2021, doi: 10.1080/09513590.2021.1958310.
22. Y. Gu, G. Zhou, F. Zhou, Q. Wu, C. Ma, Y. Zhang, J. Ding, and K. Hua, "LifeModifications and PCOS: old story but new tales," Frontiers in Endocrinology, vol. 13,article 808898, Apr. 2022, doi: 10.3389/fendo.2022.808898.
23. 23. V. De Leo, M. C. Musacchio, V. Cappelli, M. G. Massaro, G. Morgante, and F. Petraglia, "Genetic, hormonal and metabolic aspects of PCOS: an update," Reproductive Biology and Endocrinology, vol. 14, no. 38, pp. 1–17, May 2016, doi: 10.1186/s12958-016-0173-x.
24. J. Pinto, N. Cera, and D. Pignatelli, "Psychological symptoms and brain activity alterations in women with PCOS and their relation to the reduced quality of life: a narrative review," Journal of Endocrinological Investigation, vol. 47, no. 7,pp. 1599–1620, Mar. 2024, doi: 10.1007/s40618-024-02329-y.
25. L. Rajashekar, D. Krishna, and M. Patil, "Polycystic ovaries and infertility: Ourexperience," Journal of Human Reproductive Sciences, vol. 1, no. 2, pp. 65–72, Jul.–Dec. 2008, doi: 10.4103/0974-1208.38936.
26. H. Mohammad Sadeghi, I. Adeli, T. Mousavi, M. Daniali, D. Calina, A. O. Docea, S. Nikfar, A. Tsatsakis, and M. Abdollahi, "Polycystic ovary syndrome:A comprehensive review of pathogenesis, management, and drug repurposing," International Journal of Molecular Sciences, vol. 23, no. 2, article 583, pp. 1–33, Jan. 2022, doi: 10.3390/ijms23020583.
27. S. Akre, K. Sharma, S. Chakole, and M. B. Wanjari, "Recent Advances in theManagement of Polycystic Ovary Syndrome: A Review Article," Cureus,vol. 14, no. 8, p. e27689, Aug. 2022, doi: 10.7759/cureus.27689.
28. S. Cowan, S. Lim, C. Alycia, S. Pirotta, R. Thomson, M. Gibson-Helm, R. Blackmore,N. Naderpoor, C. Bennett, C. Ee, V. Rao, A. Mousa, S. Alesi, and L. Moran, "Lifestyle management in polycystic ovary syndrome – beyond diet and physicalactivity," BMC Endocrine Disorders, vol. 23, no. 14, pp. 1–33, 2023, doi: 10.1186/s12902-022-01208-y.
29. M. Zehravi, M. Maqbool, and I. Ara, "Healthy lifestyle and dietary approaches to treating polycystic ovary syndrome: A review," Open Health, vol. 3, pp. 60–65, 2022, doi: 10.1515/openhe-2022-0008.
30. M. Iervolino, E. Lepore, G. Forte, A. S. Laganà, G. Buzzaccarini, and V. Unfer, "Natural molecules in the management of polycystic ovary syndrome (PCOS):An analytical review," Nutrients, vol. 13, no. 5, p. 1677, May 2021, doi: 10.3390/nu13051677.

SJIF Impact Factor (2025): 8.688| ISI I.F. Value: 1.241| Journal DOI: 10.36713/epra2016 ISSN: 2455-7838(Online)
EPRA International Journal of Research and Development (IJRD)
Volume: 10 | Issue: 12 | December 2025 - Peer Reviewed Journal

31. *S. S. Merkin, J. L. Phy, C. K. Sites, and D. Yang, "Environmental determinants of polycystic ovary syndrome," Fertility and Sterility, vol. 106, no. 1, pp. 16–24,Jul. 2016, doi: 10.1016/j.fertnstert.2016.05.011.*
32. *A. Wal, P. Wal, N. Saraswat, and S. Wadhwa, "A detailed review on herbal treatments for treatment of PCOS – Polycystic ovary syndrome (PCOS)," Current Nutraceuticals, vol. 2, no. 3, pp. 192–202, 2021, doi: 10.2174/2665978602666210805092103.*
33. *A. Bargiota and E. Diamanti-Kandarakis, "The effects of old, new and emerging medicines on metabolic aberrations in PCOS," Therapeutic Advances in Endocrinology and Metabolism, vol. 3, no. 1, pp. 27–47, Feb. 2012, doi: 10.1177/2042018812437355.*
34. *S. H. Oguz and B. O. Yildiz, "An update on contraception in polycystic ovary syndrome," Endocrinology and Metabolism, vol. 36, no. 2, pp. 296–311, Jun. 2021,doi: 10.3803/EnM.2021.958.*
35. *M. Amiri, F. Nahidi, R. B. Yarandi, D. Khalili, M. Tohidi, and F. R. Tehrani, "Effects of oral contraceptives on the quality of life of women with polycystic ovary syndrome: a crossover randomized controlled trial," Health and Quality of Life Outcomes, vol. 18, no. 293, pp. 1–12, Aug. 2020, doi: 10.1186/s12955-020-01544-4.*